HEAD INJURY WITH SUBSEQUENT, INTERMITTENT, NONSCHIZOPHRENIC, PSYCHOTIC SYMPTOMS AND VIOLENCE

Carl C. Bell, MD, and Ruby P. Kelly, MSW Chicago, Illinois

A young, black, adult woman presented to an outpatient clinic for treatment with a history of intermittent, nonschizophrenic, psychotic symptoms. Blacks, because of their situational sociology, may be more predisposed to severe head injuries, and this acquired biologic factor may be, in part, responsible for the high rates of black-on-black murder. The use of beta blockers is discussed as an adjunct in the treatment of violence occurring in patients with a past history of severe head injury.

A young, black, adult woman presented to an outpatient clinic for treatment with a history of intermittent, nonschizophrenic, psychotic symptoms. Her post-psychotic, psychosocial functioning lacked the significant deterioration found in the post-psychotic adjustment of schizophrenics; however, it was characterized by irritability, an inability to get along with significant others, and a tendency to have intermittent, explosive episodes of violence. The patient's past history revealed her intermittent, nonschizophrenic, psychotic symptoms, and violent behavior began subsequent to a severe head injury.

Requests for reprints should be addressed to Dr. Carl C. Bell, Community Mental Health Council, 8704 S. Constance, Chicago, IL 60617.

CASE HISTORY

The patient was born in the early 1960s following an essentially normal gestation and delivery. She was considered an average baby who had achieved all developmental milestones within the normal range. Her relationship with family members during her early development was described as affectionate, outgoing, and playful. Her biological parents separated when the patient was aged 3 years, but the patient was not reported to have manifested any separation issues as a result. Overall, the patient's early developmental years were nonsignificant for poor frustration tolerance, temper outbursts, or violent tendencies. Her mother remarried while the patient was in kindergarten, and she and her stepfather developed a close relationship. Because the patient was the youngest of three girls, it was reported that her stepfather tended to be a bit indulgent with her. Her relationship with her mother and eldest sister was close with elements of respect and identification, but her relationship with her next older sister was competitive for the mother's attention. However, their sibling rivalry was not considered unusual.

The patient's academic standing throughout elementary school was slightly below average. Socially, however, she was very outgoing, had lots of friends, and had good relations with her teachers. She was active in sports, cheerleading, dancing, and other activities. There were no reports of truancy, fights, temper outbursts, or impaired relationships with others throughout her elementary school years. She grad-

uated on time, and went on to attend a local neighborhood high school, but continued to achieve slightly below average. Popularity among peers and teachers continued, and she participated in various organizations, sports, cheerleading, and dancing activities. Her family relations continued as they had through childhood, and the patient began heterosexual relationships by dating a high school sweetheart off and on for four years. During her late adolescence, the patient experienced a traumatic event that resulted in a significant change.

When the patient was a senior in high school, she and her best friend went on a double date with their boyfriends. All of them had been drinking and subsequently were involved in a tragic auto accident in which everyone was killed except the patient. She sustained a head injury and was comatose for ten days. After recuperating from the accident, she returned to high school, but this proved to be difficult, as she encountered too many associations that reminded her of her deceased friends. She began to withdraw, and imagined she would see her deceased friends on the school grounds. Her mother transferred her to another area high school, where the patient completed her high school requirements, although she continued to withdraw and failed to make any new friends. During the six months after the accident, the family noticed several behavioral changes; the patient was abrasive toward others, she had paranoid thoughts that others were "out to get her," she evidenced difficulty in concentrating, and had crying spells. This was in sharp contrast to her behavior before the accident, which was described as cheerful, playful, active, competitive, affectionate, easy to get along with, outgoing, and charismatic. Despite these behavioral changes, the family was not overly alarmed, as they felt the patient was going through a normal grieving process. After her high school graduation in 1980, the patient was employed as a waitress in a fast-food restaurant, but was terminated after a three-week period because of her inability to get along with others.

The patient next attended a beauty culture school for six months, but, again, her success was curtailed because of her inability to get along with the teacher. She was also terminated from her next employment in a clothing store because she generated interpersonal friction. During this year after graduation, the family noticed additional behavioral changes: rages, temper outbursts, slamming doors, physical and verbal confrontations with others, and suicidal ideation.

During the patient's young adult years, her level of functioning became increasingly worse. Although motivated to seek gainful employment, she would seldom be able to stick with a vocation because of her poor frustration tolerance and equally poor impulse control. In 1982, she successfully completed a four-month program at a local vocational training center, and received a certificate in medical technology. During this same year she worked in two other blue collar jobs that ended in termination because of her inability to get along with supervisors and coworkers. Her sporadic temper outbursts and impaired relations with others continued until the early months of 1984. At this point, the family became more concerned over the patient's constant complaint of boredom, increased withdrawn behavior, excessive sleeping pattern, destruction of property, frustrations regarding her lack of employment, and inability to move out on her own. As a result, in June 1984, the family sought help from a local city mental health clinic (the patient was aged 23 years—seven years after the accident).

The family's main motivation for seeking help at this time was that the patient had become "unmanageable." The patient was diagnosed as being schizophrenic, paranoid type, and she was placed on oral fluphenazine, but was later switched to fluphenazine decanoate injections. Her mother stated that her daughter's behavior improved with this treatment; however, the patient became noncompliant, as she complained that the medication slowed her down and made her feel "like a mummy." In September 1984, the mother had the patient admitted to a private university psychiatric hospital because the patient had spent one week in bed, was irritable, and exhibited poor concentration.

Upon admission to the hospital, the patient underwent a thorough physical examination, laboratory tests, and psychiatric evaluation. The physical examination was within normal limits. The neurologic examination showed the patient to be alert and oriented to person and place, but not to time. Her cranial nerves were intact; her motor functions were appropriately strong and symmetrical bilaterally; and her sensory functions were intact regarding touch, temperature, and pain. She revealed a normal capacity for cerebellar coordination on the right, as she was able to successfully complete the finger-to-nose and heel-to-shin tests on that side; however, there was some awkwardness on the left side. Her gait was nor-

mal, but the presence of Romberg's sign was questionable, because the patient fell asleep when she closed her eyes. Routine laboratory results were all within normal limits. The electroencephalogram results showed a normal tracing while the patient was in the awake and drowsy state, and there were no focal paroxysmal or epileptiform patterns seen. However, nasal leads were not recorded.

The psychiatric evaluation revealed anxious behavior, restlessness, incoherent speech, flight of ideas, and loosening of associations. Her mood was sad and constricted. She reported a ringing sound of bells and other noises in her ears and paranoid ideation. She also evidenced impaired memory. Initially, the diagnosis was questionable: post-traumatic stress disorder, schizophrenia, major depressive episode with psychotic features, and possibly an organic affective disorder were all considered. She was hospitalized for six weeks and was placed in a structured day program, which included occupational therapy, music therapy, dance therapy, multiple family meetings, small verbal groups, and individual therapy. During her hospitalization, the patient continued to exhibit psychotic symptoms and later admitted to infrequent auditory hallucinations.

As a result of the diagnostic uncertainty, she was given psychological tests (the Holtzman Ink Blot and Sentence Completion tests) that indicated slightly abnormal thought processes. The Minnesota Multiphasic Personality Inventory (MMPI) was also administered and the patient was found to have characteristics compatible with a schizophrenic disorder. At discharge, she was diagnosed as schizophrenic, disorganized type. Her aftercare plans included medication management (trifluoperazine, 20 mg at bedtime, and benztropine, 2 mg, if needed) and followup at an outpatient day treatment program. She failed to comply with the aftercare plan.

After discharge, the patient was able to function without psychotic symptoms for over a year, although she was not on medication. During this period, however, she continued to evidence sporadic temper outbursts and impaired relations with others. In the fall of 1985 (approximately the anniversary date of her accident), her family noted an exacerbation of depressive symptoms. These symptoms persisted for several months, after which the patient began to develop signs of agitation that progressively worsened. In the summer of 1986, she was rehospitalized at a state psychiatric hospital for the following reasons:

(1) disrupting the household, (2) easily enraged, (3) angry all of the time due to feelings that everyone was plotting against her, (4) a fight with her next older sister, in which the patient pushed her sister down a flight of stairs, and (5) throwing firecrackers at children who were playing downstairs. She was hospitalized for two weeks and was managed on haloperidol, 5 mg three times a day, and benztropine, 2 mg twice a day. She was discharged earlier than anticipated because she did not return to the hospital after she had been issued a pass. Her discharge plan was to participate in a structured day-treatment program and vocational training.

The patient followed the recommendation for aftercare treatment at the Community Mental Health Council. The psychiatric evaluations completed in August 1986 revealed most of the previously reported history, and an evaluation of the patient's current mental status was as follows: the patient was appropriately and neatly dressed, and behaved in an uncooperative and hyperactive fashion. Her speech was characterized by confusion, circumstantiality, and pressure. Her affect was constricted in range and representative of a dysphoric mood. She denied current homicidal or suicidal ideation and auditory or visual hallucinations. She was oriented to person and place, but marginally oriented to time. Her recent memory was fair for events of the day, and her ability to differentiate the current mayor of Chicago from the current president of the country was initially poor. But she was able to correct herself with prompting and more labored concentration. Her mathematics skills were below what one would have expected from a high school graduate, which seemed to indicate poor concentration. The patient demonstrated an instability of affect because of a tendency to switch from a sullen, dysphoric affect to unreasonable anger, and this instability decreased the patient's ability to maintain age-appropriate social judgment.

Diagnostic formulation described the patient as a 25-year-old, single, black woman who had a history of crying spells, fluctuating sleep patterns, violent outbursts, destruction of property, verbal and physical confrontation with others, suspiciousness, paranoid ideation, poor sensorium and cognition skills, and two episodes of psychiatric hospitalizations due to intermittent, nonschizophrenic, psychotic symptoms. These behaviors became manifest six months following an accident in which the patient sustained a head injury and was comatose for ten days. Before the ac-

cident she was functioning at a perceptually higher level of psychosocial functioning. Based upon this formulation, the diagnoses of organic affective disorder and intermittent explosive disorder were assigned. Initially, the patient was placed on loxapine, 25 mg, and benztropine, 2 mg twice a day, but because the patient complained of excessive fatigue, her medication was gradually reduced to the current level of loxapine, 10 mg at bedtime. She was also placed on propranolol, the dose of which was gradually being increased while her loxapine dose was being decreased, until a final dose of propranolol of 40 mg twice a day was reached.

The patient is currently compliant with treatment, and her affective disorder with psychotic symptoms (presumably due to an organic cause) and her intermittent explosive behaviors have ceased. While her relationships with significant others have improved markedly, she continues to show subtle sensorium and cognitive deficiencies that hamper her adjustment, and she maintains a tendency to deny her difficulties, although she is much more satisfied with her current outpatient treatment.

DISCUSSION

This case illustrates how a head injury causing a coma may later result in an organic affective disorder with psychotic symptoms and an organic personality disorder-explosive type (referred to in DSM-III¹ as an intermittent explosive disorder). The subject of organic affective disorders with psychotic symptoms has been discussed elsewhere, 2-4 and will not be extensively discussed here, except as the problem relates to blacks. Bell et al⁵ demonstrated a substantial proportion of blacks with prior psychotic episodes reported a history of having had an episode of coma. Over one half of the patients studied reported having had a coma, with 15 percent of this aftercare sample reporting their unconsciousness lasted longer than 24 hours. These findings imply that a significant proportion of psychotic illness in blacks may be related to a prior head injury. Another point of interest was that the patient was diagnosed as schizophrenic based on psychiatric evaluation and psychological testing (MMPI). The issues of misdiagnosis of blacks with affective disorders⁶⁻⁹ and the excessive diagnosis of blacks as being schizophrenic based on psychological testing are on record, 10,11 but the question of how a substantial prevalence of head injury in blacks obscures diagnostic assessments has yet to be investigated.

Organic personality disorder-explosive type is characterized by a persistent pattern of behavior with recurrent outbursts of aggression or rage often resulting in serious assault or destruction of property. According to revised DSM-III criteria, the explosive behavior is grossly out of proportion to any precipitating psychosocial stressor, and is not due to psychotic symptoms, major affective disorders, or clouding of consciousness. Unfortunately, these revised criteria do not clearly recognize that head injury may also cause intermittent, psychotic, nonschizophrenic symptoms. These exclusion criteria (in the authors' opinion) should refer only to clearly "functional" psychotic or affective symptoms, and not to psychotic or affective symptoms that seem to be related to an organic cause. Patients, such as the reported case, who have intermittent, psychotic, nonschizophrenic symptoms secondary to head trauma may need treatment with low doses of antipsychotic medication, but this should not preclude the recognition of their explosive disorders. The revised DSM-III criteria also require a history of unconsciousness for at least 15 minutes following a head injury, seizures, drug or alcohol use, infection, or physical illness; or evidence of an abnormality of brain function or structure (eg, history of seizure disorder, neurologic "soft signs," or history of an abnormal electroencephalogram), which the patient exhibited.

A recent review of the literature^{12,13} documents some of the research that led to the establishment of the understanding that head injury may have some significance in the origin of violent behavior and is recommended for further study. Lewis et al¹⁴ have recently furthered this understanding by their work on the neuropsychiatric impairment of death row inmates who had murdered. All of the individuals studied had a history of significant head injury, one third had evidence of major neurologic impairment, and an additional one half had less serious neurologic problems that were identifiable by neurologic examination.

The understanding that head injury may have some causative significance for violent behavior has particular relevance for blacks. Several studies have shown that blacks are more at risk for such head injury. Whitman et al¹⁵ found the head trauma rate for blacks living in the suburbs was 394 per 100,000, and head trauma rates for inner-city blacks was 403 per

100,000. The head injury rate for suburban whites was 296 per 100,000. In their study, falls were the most common cause of head injury for inner-city black children aged under 16 years. Rivara and Mueller¹⁶ found that for ". . . all races, injury death rates [from head trauma] are inversely related to income level, and much of the difference seen in death rates between racial groups may be the result of existing discrepancies in income." Further, the incidence of head injury among nonwhites has been shown to be nearly 50 percent higher than among whites, and the male-to-female ratio is slightly higher among nonwhites as well.

That blacks have a greater incidence of head injury, and that head injury has been associated with causing a predisposition to violence, casts significant doubts on the "subculture of violence" explanation that has been expounded concerning the high rates of blackon-black murder. Clearly, the high rates of black-onblack violence may be more due to the high incidence of head injuries found in poor neighborhoods than to a supposed "subculture" existing in those neighborhoods. Thus, it has been proposed that physicians pay attention to the situational sociologic conditions that predispose blacks to head injury (eg, inadequate housing that leads to a disproportionate number of free falls from heights among black and Hispanic children¹⁷) and help reduce those situational sociologic conditions rather than address the "red herring" of subcultural shortcomings of black people. 18,19 Further, it has been suggested that, in addition to reducing the acquired biologic factors contributing to violence, physicians and health professionals address the situational sociologic conditions that also cause psychological factors (eg, self hatred) that predispose blacks to violence.^{20,21} Physicians and health professionals can also directly repair those psychological factors that predispose blacks to violence.²²

It has been suggested that physicians directly treat those acquired biologic conditions leading to violence by beginning to study thoroughly the various medications that seem to have promise in reducing violence that partly stems from acquired biologic factors such as head injury. Propranolol, which has shown a significant promise in this regard, was used in the study patient with good results. This medication has been shown to be useful in reducing violence in several studies, ¹³ but the bulk of the subjects in these studies were white.

Because propranolol has been reported to be less

effective in the treatment of black hypertensives, questions have been raised about propranolol's efficacy in blacks who are explosive. Schorer²³ noted, "too few black patients have been included in studies [on explosiveness] to reach any conclusions about the efficacy of propranolol, or whether race is a differentiating factor." The lack of antihypertensive effectiveness in blacks taking propranolol may be unrelated to the effect it may have on reducing violent behavior. Should propranolol be useful in treating intermittent explosive disorders in blacks (as has been the authors' experience), a lack of antihypertensive effects may be a boon in disguise, as a physician treating a black patient for these disorders would not need to be concerned about cardiovascular side effects of the medication. More studies are urgently needed to elucidate these important questions, as it is clear that the answers would greatly benefit blacks.

It is hoped that this case history will give physicians some motivation to present their own case studies or to embark upon more controlled clinical research to test the hypothesis that propranolol may be useful in reducing the intermittent explosive violence that results from head injury, and head injury may play a significant role in the high rates of black-on-black murder.

Acknowledgment

Judy Woods prepared the manuscript.

Literature Cited

- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, ed 3. Washington, DC: American Psychiatric Association, 1979.
- 2. Shukla S, Cook BL, Mukherjee S, et al. Mania following head trauma. Am J Psychiatry 1987; 144:93-96.
- Riess H, Schwartz CE, Klerman GL. Manic syndrome following head injury: Another form of secondary mania. J Clin Psychiatry 1987; 48:29–30.
- 4. Krauthammer C, Klerman GL. Secondary mania. Arch Gen Psychiatry 1978; 35:1333–1339.
- Bell CC, Thompson B, Shorter-Gooden K, et al. Prevalence of coma in black subjects. J Natl Med Assoc 1985; 77:391–395.
- Bell CC, Mehta H. Misdiagnosis of black patients with manic depressive illness. J Natl Med Assoc 1980; 72:141–145.
- Bell CC, Mehta H. Misdiagnosis of black patients with manic depressive illness: Second in a series. J Natl Med Assoc 1981; 73:101–107.
- 8. Jones BE, Gray BA, Parson EB. Manic-depressive illness among poor urban blacks. Am J Psychiatry 1981; 138:654–657.
- Mukherjee S, Shukla S, Woodle J, et al. Misdiagnosis of schizophrenia in bipolar patients: A multiethnic comparison. Am J Psychiatry 1983; 140:1571–1574.
- 10. Adebimpe VR, Gigandet J, Harris E. MMPI diagnosis of black psychiatric patients. Am J Psychiatry 1979; 136:85–87.

- 11. Williams CL. Issues surrounding psychological testing of minority patients. Hosp Comm Psychiatry 1987; 38:184–189.
- 12. Bell CC. Coma and the etiology of violence, part 1. J Natl Med Assoc 1986; 78:1167–1176.
- 13. Bell CC. Coma and the etiology of violence, part 2. J Natl Med Assoc 1987: 79:79–85.
- 14. Lewis DO, Pincus JH, Feldman M, et al. Psychiatric, neurological, and psychoeducational characteristics of 15 death row inmates in the United States. Am J Psychiatry 1986; 143:838–845.
- 15. Whitman S, Coonley-Hoganson R, Desai BT. Comparative head trauma experience in two socio-economically different Chicago-area communities: A population study. Am J Epidemiology 1984: 119:570–580.
- 16. Rivara FP, Mueller BA. The epidemiology and prevention of pediatric head injury. J Head Trauma Rehabil 1986; 1:7–15.

- 17. Ramos SM, Delany HM. Free falls from heights: A persistent urban problem. J Natl Med Assoc 1986; 78:111–115.
- 18. Hawkins DF, ed. Homicide Among Black Americans. Lanham, Md, University Press of America, 1986.
- 19. Bell CC, Prothrow-Stith D, Smallwood-Murchinson C. Black-on-black homicide: The National Medical Association's responsibilities. J Natl Med Assoc 1986; 78:1139–1141.
- 20. Poussaint A. Why Blacks Kill Blacks. Virplanck, NY: Emmerson-Hall, 1972.
- 21. Bell CC. Preventing violence, editorial. New Physician 1986; 35:7–8.
- 22. Thomas CW. Pride and purpose as antidotes to black homicidal violence. J Natl Med Assoc 1987; 79:155–160.
- 23. Schorer CE. Behavioral efficacy of propranolol in black patients. J Natl Med Assoc 1987; 79:221–222.